when I met with my first case in 1907. Dr. Sullivan's article had not then been published, so I improvised a means of reconstructing a common duct which had been injured in the preceding operation. The patient, who suffered two or three years with icterus whenever his fistula closed, but was relieved when it was open, desired something to be done. It was possible to find a dilated proximal common duct. The gallbladder had been removed at a former operation. Into this dilated common duct it was possible to introduce a rubber tube. There was no possibility in the cicatricial tissue which surrounded this field (he had had three previous operations) of finding the distal portion of it. So a pursestring suture was made in the duodenum at the point most closely lying to the proximal end of the common duct, and a tube introduced through an incision in the center of this area after a pursestring suture had been made in the common duct proximal end and a tube inserted. Reasoning that it was essential that that tube should come away not at once, but after some time, I did this: I turned back a cuff on the rubber tube about one-quarter of an inch, then turned that back again so that I had a round knob on the end of the tube about twice the size of the tube, narrowing its lumen, but permitting the bile to flow. With the proximal end in the common duct, it was easy to constrict the common duct, leaving its mucosa attached so that it brought it close to the duodenum. I wanted the tube to come away, but not too soon. I made it 8 inches long and let the 8 inches go down the duodenum, so that the constant peristaltic pulling for five or six weeks all that time gave a chance for epithelium to develop. That scheme works. I have done it now for the fourth time, and one of my associates, calling me to assist him in a difficult icteric case, repeated it successfully for the fifth time. The practical hint is, to follow the suggestion of Willy Meyer, bring the duodenum as close as possible to the stump of the proximal end of the common duct and keep it patulous until the mucosa can grow to mucosa. While the experience of any of us will not be great in numbers, so simple a resource as that described can be found in any operating room.

Dr. I. J. Strauss, Chicago: In 1912, when I reconstructed ureters from the fascia lata, I got a patent ureter. Epithelium will grow on any medium. You cannot prevent epithelium from growing over a hollow viscus. As to end results, experimentally from the bile duct work, success is due entirely to technical skill. If you take a fascial transplant uppermost, then the hydrostatic bile will distend the tube. Dr. Davis is correct. When I did ureteral work I did bile operations and put a stone into the duct. Then the hydrostatic action caused stricture, and you take out the stone and you have no stricture whatever. This shows that the bile within the common duct is under a very low pressure, and consequently there is always a constriction. If we have high hydrostatic action, we would have no pressure, and these cases of reconstruction of the bile duct would be a success.

Dr. J. Shelton Horsley, Richmond, Va.: The pathology of a biliary fistulous tract is intimately connected with such reconstructive work. Of course, the fistulas that do not close impress us. What is the proportion of external biliary fistulas that do not close to those that do? Exceedingly small. If they do not close we know the reason; usually some obstruction to the bile lower down. The Sullivan operation is as much a transplantation as when a vein or fascia is used and has the advantage of a pedicle for bland supply. Sullivan's procedure has the additional advantage, too, of using tissue in the neighborhood, which has some immunity to the irritating effects of the biliary discharges; but a free transplant is not so well nourished, has no immunity to the irritating discharges, and will eventually contract. The ideal method is to unite the stump of the duct to the submucosa of the duodenum. Dr. Sullivan has authorized me to say that he is in no way responsible for the T-tube. In pulling it out it causes too much damage.

Teach Health to the Children.—Since 90 per cent. of our American children leave school at or before the eighth grade, public health must be taught in the grades if the majority of the population is to be reached.—*Minnesota Public Health Association Journal.*

HEMOPHILIA

EXPERIMENTAL DATA BEARING ON THE EFFECT OF GLYCERINIZED EXTRACTS OF VISCERAL HEMOPHILIC TISSUE ON THE COAGULATION TIME OF BLOOD *

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AND

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PHILADELPHIA

Inasmuch as the active etiology of hemophilia remains one of the unsolved mysteries of medicine, and inasmuch as medical science affords us no remedial agent which will permanently eradicate the disease, the investigations to be recorded were undertaken with the hope that some new information with reference to the cause of the remarkably slow coagulative property of the blood in hemophilia might be provided. The opportunity to conduct these investigations came to us by reason of the death of a 6 year old lad, who presented the classical family history of this disheartening affection. He had been admitted to the pediatric service of the Mount Sinai Hospital of Philadelphia on several occasions. Each time profuse hemorrhage had followed the slightest trauma to skin or mucosa. Usually he bled from the tongue, mouth or lips—the result of teeth bites from falls. The slightest pressure would produce extensive subcutaneous ecchymoses. At no time would the bleeding cease spontaneously or by the use of ordinary measures. Horse serum or the direct injection subcutaneously of freshly drawn human blood was always necessary to control the bleeding. The anemia following one of these attacks was always profound, and frequently he would become highly toxic following an injection, presenting hyperpyrexia, delirium, urticaria and other evidences of the intracirculatory destruction of foreign protein. Once the bleeding ceased, however, his color and animation would return with amazing rapidity. His father did not bleed, nor did his mother, nor his sister, but his brother, who even now is a patient under the care of one of us (Lowenburg) at the Mount Sinai Hospital, just recovering from a severe hemorrhage following a slight trauma on the forehead, and which required intravenous transfusion to control it, frequently has attacks of bleeding. There is no history of consanguinity. His maternal uncle is a bleeder, having required intravenous transfusion to control what appeared to be spontaneous hematuria. This occurred on two occasions and almost cost the patient his life. Another maternal uncle bled to death. Thus it is seen that we are dealing with a typically classical case of hemophilia. Our patient died at the Mount Sinai Hospital following uncontrollable intestinal hemorrhage of unknown origin.

Assuming that the blood of hemophiliacs does not come in contact with extravascular tissues (skin, muscle, fascia, etc.), that the coagulation time under such circumstances is unduly prolonged beyond normal is not the subject of professional controversy. Howell's experiment, wherein he showed that hemophilic blood drawn directly into a test tube sometimes consumes five hours in clotting, may be readily verified by repetition. Normal blood is coagulated within from thirty

* Read before the Section on Diseases of Children at the Sixty-Ninth Annual Session of the American Medical Association, Chicago, June, 1918.

to forty minutes. Hemophilic blood presents no striking abnormal variations in the white and the red cell count and the blood plaques. Various authorities have, however, proposed divergent hypotheses as to the elemental causal factor, some ascribing it to an overabundance of anticoagulative substances and others to a deficiency of procoagulative agents.

Thus Morawitz, as well as Lossen,[1] attributed the defect to a lack of thrombokinase; Weil[2] attributed it to an excess of antithrombin; Wright, to calcium deficiency, while Addis[3] and Howell[4] showed experimentally that there was a lack of prothrombin. Howell, as well as Nolf,[5] found no calcium deficiency, and indeed Lossen proved that adding calcium to hemophilic blood causes no decrease in coagulation time. Howell, furthermore, demonstrated no deviation from a normal fibrinogen content and that the blood of a hemophilic, when brought in contact with the tissues of the same person, is coagulated rapidly. For example, if during venipuncture of a hemophiliac, the technic is faulty, and some blood traverses the subcutaneous tissues before it is drawn into a test tube, the blood will clot in little longer time than normal.

Since all workers are practically agreed that extracts of the external tissues exert a marked influence in diminishing the coagulation time of hemophilic blood, it occurred to us to investigate the effects of extracts or emulsions of the various internal tissues, both from our dead hemophilic patient, as well as from those of an apparently healthy, normal boy who died by accident, and to ascertain the effect of these, if any, on the coagulation time of blood.

Our work consisted in preserving samples of the following tissues: brain, thyroid, heart, liver, kidney, suprarenal, pancreas, spleen, muscle and bone marrow.

PREPARATION OF TISSUES

The tissues were cut into very small pieces, and washed in running water for about three hours to rid them of as much blood as possible. They were then ground in a mortar with three times their volume of physiologic sodium chlorid solution and a little glycerin was then added.

To test the effect of these tissue extracts on coagulation of blood, advantage was taken of the method outlined by Howell, consisting of first, oxalating blood plasma and then reactivating coagulation in such plasma by the addition of a suitable amount of calcium chlorid. In this way normal blood plasma clots uniformly in from eight to twelve minutes. The effect of the addition of the various tissue extracts to such preparations of plasma was easily ascertained by gently tilting the test tubes and observing closely how long it took the plasma to form a firm coagulum.

Blood from a normal person was drawn by venipuncture into a Lüer syringe and at once run into a test tube containing a solution of sodium oxalate in physiologic sodium chlorid solution, so that the oxalate was present in about 0.12 per cent. of the entire volume. The oxalated blood was then centrifuged and the supernatant yellowish green plasma pipetted off. The clotting of the plasma was reactivated by adding calcium chlorid as follows: 0.5 c.c. of oxalated plasma was placed in each of five test tubes, and to these were added in series of 0.1, 0.2, 0.3, 0.4, 0.5 c.c. of 0.5 per cent. solution of calcium chlorid, and the coagulation time observed in each test tube to ascertain which amount of calcium chlorid caused the quickest and most uniform coagulum. After several repetitions of this test 0.35 c.c. were found to be most efficient, and with this amount the plasma clotted in eight minutes and forty seconds. Any amount less or more than 0.35 c.c. caused slow, uneven, or no coagulation at all.

Into each of three test tubes were placed 0.5 c.c. of oxalated plasma, one drop of the tissue emulsion and 0.35 c.c. of the calcium chlorid solution. The tubes were closely observed and frequently gently tilted to determine the coagulation time. This procedure was carried out with every type of tissue studied, both hemophilic and normal, together with controls of calcium and plasma alone, glycerin alone, and with thromboplastin-Squibb and that prepared in our own laboratory. These reactions were repeated several times and corresponded with the result shown in the accompanying table.

COAGULATION TIME OF PLASMA AND CALCIUM AFTER ADDITION OF TISSUE

Extracts	Hemophilic Min.	Hemophilic Sec.	Normal Min.	Normal Sec.
Brain	4		3	
Thyroid	10	30	4	
Heart	4	15	4	30
Liver	9	45	5	
Kidney	4	20	3	40
Suprarenal	4	30	3	10
Pancreas	6	30	4	
Spleen	6		5	
Muscle	5		3	15
Bone marrow	6		4	25
Controls				
Oxalated plasma	No coagulation			
Plasma and calcium			8	40
Glycerin			8	30
Thromboplastin			2	(Squibb)
			2	20(Ours)

RESULTS

It will be noted that the normal tissue extracts uniformly accelerate the coagulation time of the calcium plasma, and that most of the hemophilic tissues with the exception of thyroid and liver seem to exert almost the same influence on coagulation. Thyroid gland and liver not only caused a prolonged coagulation time, amounting to two and one-half times the corresponding normal tissues, but actually inhibited the action of the calcium added to the plasma, so that the coagulation of the calcium plasma was prolonged almost two minutes.

These observations were repeated with uniform results, and though admittedly limited to the tissues of one hemophiliac, yet they are strikingly significant, in that there is a possibility that the thyroid and even the liver may secrete an antithrombic substance or enzyme which may be in part, if not principally, the cause of the deranged coagulative mechanism of hemophilic blood. These observations are recorded with the purpose of stimulating further investigation along similar lines, hoping to localize the offending principle causing this now so-called idiopathic disease.

CONCLUSIONS

1. Not only the external tissues, but also the various other tissues of the normal body accelerate blood coagulation.

2. Hemophilic tissues, excepting thyroid and liver, have the same general effect on coagulation as normal tissues exert.

1. Morawitz and Lossen: Deutsch. Arch. f. klin. Med., 1908, **94**, 110.
2. Weil: Presse méd., 1905.
3. Addis, T.: Jour. Path. and Bacteriol., 1911, **15**, 436.
4. Howell, W. H.: The Condition of the Blood in Hemophilia, Thrombosis and Purpura, Arch. Int. Med., January, 1914, p. 76; Am. Jour. Physiol., 1912-1913, 1910.
5. Nolf, Ergebn. d. inn. Med. u. Kinderh., 1913, **10**, 275.

3. Extracts of the thyroid and of the liver of hemophiliacs markedly prolong coagulation of blood.

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ABSTRACT OF DISCUSSION

Dr. William Weston, Columbia, S. C.: In the treatment of these purpuric conditions, it may be well to keep in mind the marked effect that fresh fruit juices have on the coagulation of the blood. For instance, we know that in scorbutus fruit juices assist markedly in the coagulation of the blood.

RECENT STUDIES IN THE ANATOMY AND PHYSIOLOGY OF TENDONS

THEIR APPLICATION TO THE TECHNIC OF TENDON OPERATIONS *

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NEW YORK

My interest in tendons was first awakened in the year 1912, when, while I was acting as volunteer in the clinic of Professor Lange, the problem of preventing postoperative adhesions was assigned as an experimental study to Dr. Henze of New Haven and to me.

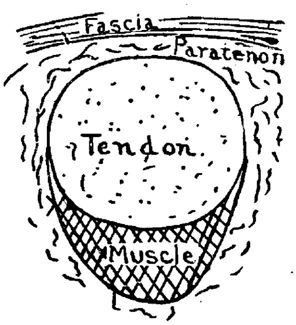

Fig. 1.— Cross-section (diagrammatic) through the tibialis anticus tendon 1 inch above the upper pole of the sheath.

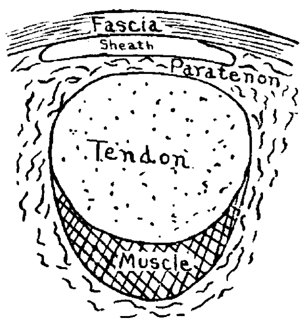

Fig. 2.— Cross-section (diagrammatic) through the tibialis anticus tendon at the level of the upper pole of the sheath.

The problem was one of great importance in Lange's eyes, since, despite his experience in 2,000 operations, the results were frequently impaired by adhesions developing subsequent to the transplantation.

It is, of course, self-evident that the function of the tendon as a means of transmitting the contraction of the muscle to the skeleton is completely inhibited by a single strong adhesion in the same way as the rope of the derrick cannot glide if clamped at a single point. In the course of our experimental investigations, which were conducted chiefly on rabbits, we utilized all manner of membrane, thin tubes of rolled silver, petrolatum, bismuth paste, fascia, peritoneum and a vein as a means of ensheathing the tendon. None of these substances, however, prevented the development of adhesions; in fact, with the exception of the Cargyle membrane, more adhesions were present after their introduction than in control experiments in which nothing was used. Finally we followed the suggestion of Biesalski and utilized the sheath of the paralyzed tendon as a physiologic pathway for the transplanted tendon; that is, one tendon was withdrawn from its sheath, cut away from the paralyzed muscle, and the substituting tendon drawn downward by means of a guide suture so as to occupy exactly the position of the original tendon. In all of the cases observed there was complete absence of adhesions, even when the limb was immobilized for thirty days subsequent to the operation.

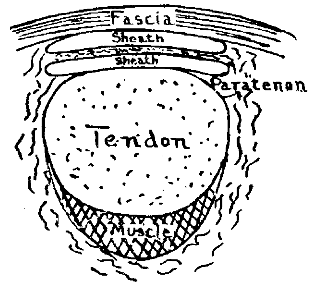

Fig. 3.— Cross-section (diagrammatic) through the tibialis anticus tendon one-half inch distal to the section shown in Figure 2.

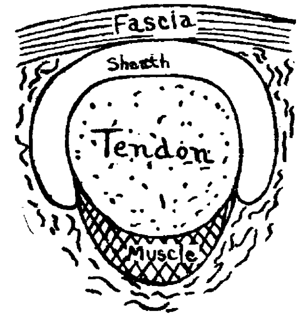

Fig. 4.— Cross-section $1\frac{1}{3}$ inches distal to the section shown in Figure 3.

This clear cut evidence in favor of Biesalski's method indicated to me the importance of coordinating the operative technic with the physiology of the structures involved. Just exactly as the normal relationship between tendon and sheath should be maintained, so, too, the normal fascia relationship, the normal tension and the normal fixation of the transplanted tendon should be made as nearly like the normal as possible. When, however, I tried to follow out this line of thought, I found that our knowledge of the physiology and anatomy of tendons was entirely inadequate for the purpose. No one had as yet considered the nature of the gliding mechanism of tendons. In no book, physiologic or surgical, had the subject of tendon tension ever been brought up for discussion. Despite the thousands of tendon operations, some of the simplest facts relative to their anatomy had never been investigated. It was necessary, therefore, before any comprehensive operative technic could be formulated, that these fundamental questions be investigated. The work was conducted by research on the cadaver, animal experimentation and observations on human beings.

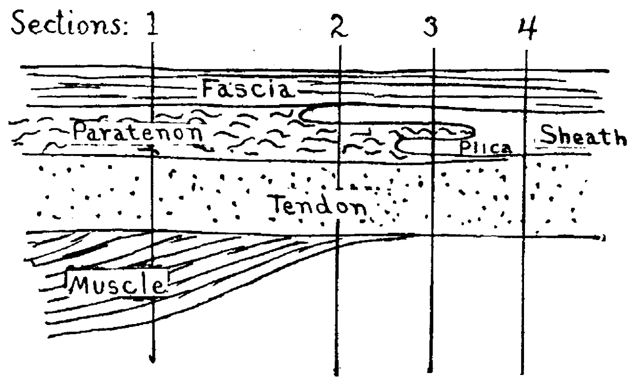

Fig. 5.—Longitudinal section (diagrammatic) of the tibialis anticus tendon, to correlate the preceding cross-sections. Note that the paratenon is prolonged downward into the sheath as a loose fold—the plica.

I can best introduce the subject of the anatomy of tendons by a series of cross-sections showing a tendon at various levels above and within its sheath. The first (Fig. 1) shows the tendon about 1 inch above the upper pole of the sheath; note that between the fascia and the tendon is a distinct gap not described

* Read before the Section on Orthopedic Surgery at the Sixty-Ninth Annual Session of the American Medical Association, Chicago, June, 1918.